L Number	Hits	Search Text	DB	Time stamp
1	3	(plasminogen adj activator) near2 (IL-2 or IL2 or (IL adj "2"))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/09 13:54
2	56	(IL-2 or IL2 or (IL adj "2")) adj inhibitor	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/09 13:54
3	13440	((IL-2 or IL2 or (IL adj "2")) adj inhibitor) near "4" (plasminogen adj activator)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/09 13:55
4	1	((IL-2 or IL2 or (IL adj "2")) adj inhibitor) near4 (plasminogen adj activator)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/09 13:55
5	1	((IL-2 or IL2 or (IL adj "2")) adj inhibitor) near10 (plasminogen adj activator)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/04/09 13:55

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TI     Proteins and nucleic acids encoding same
IN     Tchernev, Velizar T., Branford, CT, UNITED STATES
       Spytek, Kimberly A., New Haven, CT, UNITED STATES
       Zerhusen, Bryan D., Branford, CT, UNITED STATES
       Patturajan, Meera, Branford, CT, UNITED STATES
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       Colman, Steven D., Guilford, CT, UNITED STATES
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Rieger, Daniel K., Branford, CT, UNITED STATES
Burgess, Catherine E., Wethersfield, CT, UNITED STATES

PI US 2004033493 A1 20040219
AI US 2002-72012 A1 20020131 (10)
PRAI US 2001-267459P 20010208 (60)
US 2001-266975P 20010207 (60)
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US 2001-271664P 20010226 (60)

DT Utility
FS APPLICATION
LREP Ivor R. Elrifi, Ph.D., Mintz, Levin, Cohn, Ferris,, Glovsky and Popeo, P.C., One Financial Center, Boston, MA, 02111
CLMN Number of Claims: 49
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 59681
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 1
AN 2001:265240 CAPLUS
DN 134:261270
TI **Plasminogen activator** for enhancement of **IL-2 inhibitor** effects
IN Moriguchi, Akira; Furuichi, Yasuhisa; Katsuta, Kiyotaka; Maeda, Masashi; Sato, Natsuki
PA Fujisawa Pharmaceutical Co., Ltd., Japan
SO PCT Int. Appl., 21 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001024784	A2	20010412	WO 2000-JP6874	20001002
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	JP 2003510351	T2	20030318	JP 2001-527783	20001002
PRAI	AU 1999-3249	A	19991004		
	AU 2000-5643	A	20000215		
	WO 2000-JP6874	W	20001002		

AB The present invention is related to a new use of a **plasminogen activator** for increasing an effect caused by **IL-2 inhibitor** and the use of a **IL-2 inhibitor** for increasing or decreasing an effect caused by **plasminogen activator**. Thus, FK506 (Prograf) was administered i.v. by a single bolus injection through the femoral vein 2 h after occlusion of the middle cerebral artery. A tissue-type plasminogen activator (t-PA) (1 mg/kg) was administered i.v. by a bolus injection followed by infusion for 30 min through the femoral vein 2 h after occlusion of the MCA. When drugs were administered 2 h after occlusion of the MCA, FK506 or t-PA showed a relatively small tendency of the inhibition of brain damage. However, the combination of FK506 and t-PA caused the significant reduction of ischemic brain damage and its inhibition is more than 23%, which is greater than that of FK506 or t-PA alone.

=> d l4 1-5 bib ab

L4 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:265240 CAPLUS
DN 134:261270
TI **Plasminogen activator** for enhancement of **IL-2 inhibitor** effects
IN Moriguchi, Akira; Furuichi, Yasuhisa; Katsuta, Kiyotaka; Maeda, Masashi; Sato, Natsuki
PA Fujisawa Pharmaceutical Co., Ltd., Japan
SO PCT Int. Appl., 21 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

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	AU 2000-5643	A	20000215		
	WO 2000-JP6874	W	20001002		

AB The present invention is related to a new use of a **plasminogen activator** for increasing an effect caused by **IL-2 inhibitor** and the use of a **IL-2 inhibitor** for increasing or decreasing an effect caused by **plasminogen activator**. Thus, FK506 (Prograf) was administered i.v. by a single bolus injection through the femoral vein 2 h after occlusion of the middle cerebral artery. A tissue-type plasminogen activator (t-PA) (1 mg/kg) was administered i.v. by a bolus injection followed by infusion for 30 min through the femoral vein 2 h after occlusion of the MCA. When drugs were administered 2 h after occlusion of the MCA, FK506 or t-PA showed a relatively small tendency of the inhibition of brain damage. However, the combination of FK506 and t-PA caused the significant reduction of ischemic brain damage and its inhibition is more than 23%, which is greater than that of FK506 or t-PA alone.

L4 ANSWER 2 OF 5 USPATFULL on STN
AN 2004:44501 USPATFULL
TI Proteins and nucleic acids encoding same
IN Tchernev, Velizar T., Branford, CT, UNITED STATES
Spytek, Kimberly A., New Haven, CT, UNITED STATES
Zerhusen, Bryan D., Branford, CT, UNITED STATES
Patturajan, Meera, Branford, CT, UNITED STATES
Shimkets, Richard A., West Haven, CT, UNITED STATES
Li, Li, Branford, CT, UNITED STATES
Gangolli, Esha A., Madison, CT, UNITED STATES
Padigaru, Muralidhara, Branford, CT, UNITED STATES
Anderson, David W., Branford, CT, UNITED STATES
Rastelli, Luca, Guilford, CT, UNITED STATES
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Gusev, Vladimir Y., UNITED STATES
Colman, Steven D., Guilford, CT, UNITED STATES
Wolenc, Adam Ryan, New Haven, CT, UNITED STATES

Pena, Carol E. A., Guilford, CT, UNITED STATES
Furtak, Katarzyna, Anosia, CT, UNITED STATES
Grosse, William M., Bransford, CT, UNITED STATES
Alsobrook, John P., II, Madison, CT, UNITED STATES
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Rieger, Daniel K., Branford, CT, UNITED STATES
Burgess, Catherine E., Wethersfield, CT, UNITED STATES

PI US 2004033493 A1 20040219
AI US 2002-72012 A1 20020131 (10)
PRAI US 2001-267459P 20010208 (60)
US 2001-266975P 20010207 (60)
US 2001-267057P 20010207 (60)
US 2001-266767P 20010205 (60)
US 2001-266406P 20010202 (60)
US 2001-265395P 20010131 (60)
US 2001-265412P 20010131 (60)
US 2001-265517P 20010131 (60)
US 2001-265514P 20010131 (60)
US 2001-267823P 20010209 (60)
US 2001-268974P 20010215 (60)
US 2001-271855P 20010227 (60)
US 2001-271839P 20010227 (60)
US 2001-273046P 20010302 (60)
US 2001-272788P 20010302 (60)
US 2001-275989P 20010314 (60)
US 2001-275925P 20010314 (60)
US 2001-275947P 20010314 (60)
US 2001-275950P 20010314 (60)
US 2001-276450P 20010315 (60)
US 2001-276448P 20010315 (60)
US 2001-276397P 20010316 (60)
US 2001-276768P 20010316 (60)
US 2001-278652P 20010320 (60)
US 2001-278775P 20010326 (60)
US 2001-278778P 20010326 (60)
US 2001-279882P 20010329 (60)
US 2001-279884P 20010329 (60)
US 2001-280147P 20010330 (60)
US 2001-283083P 20010411 (60)
US 2001-282992P 20010411 (60)
US 2001-285133P 20010420 (60)
US 2001-285749P 20010423 (60)
US 2001-288327P 20010503 (60)
US 2001-288504P 20010503 (60)
US 2001-294047P 20010529 (60)
US 2001-294473P 20010530 (60)
US 2001-296964P 20010608 (60)
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US 2001-299324P 20010619 (60)
US 2001-312020P 20010813 (60)
US 2001-312908P 20010816 (60)
US 2001-312889P 20010816 (60)
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US 2001-315470P 20010828 (60)
US 2001-316447P 20010831 (60)
US 2001-318115P 20010907 (60)
US 2001-318118P 20010907 (60)
US 2001-318740P 20010912 (60)
US 2001-323379P 20010919 (60)
US 2001-330308P 20011018 (60)
US 2001-330245P 20011018 (60)
US 2001-332701P 20011114 (60)
US 2001-271664P 20010226 (60)
DT Utility

FS APPLICATION
LREP Ivor R. Elrifi, Ph.D., Mintz, Levin, Cohn, Ferris,, Glovsky and Popeo, P.C., One Financial Center, Boston, MA, 02111
CLMN Number of Claims: 49
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 59681
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 5 OF 5 WPINDEX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2001-354885 [37] WPINDEX
DNC C2001-109869
TI Use of a plasminogen activator for increasing an effect caused by interleukin-2 inhibitor and for treating acute or chronic cerebral neurodegenerative diseases, e.g. cerebral ischemic diseases and/or brain damage caused by ischemia.
DC B04 D16
IN FURUICHI, Y; KATSUTA, K; MAEDA, M; MORIGUCHI, A; SATO, N
PA (FUJI) FUJISAWA PHARM CO LTD
CYC 21
PI WO 2001024784 A2 20010412 (200137)* EN 21p
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: JP US
EP 1223969 A2 20020724 (200256) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 2003510351 W 20030318 (200321) 25p
ADT WO 2001024784 A2 WO 2000-JP6874 20001002; EP 1223969 A2 EP 2000-963073 20001002, WO 2000-JP6874 20001002; JP 2003510351 W WO 2000-JP6874 20001002, JP 2001-527783 20001002
FDT EP 1223969 A2 Based on WO 2001024784; JP 2003510351 W Based on WO 2001024784
PRAI AU 2000-5643 20000215; AU 1999-3249 19991004
AB WO 200124784 A UPAB: 20010704
NOVELTY - Use of a plasminogen activator for manufacturing a medicament for increasing an effect caused by an IL-2 (interleukin-2) inhibitor, e.g. tacrolimus or its hydrate or a cyclosporin.

DETAILED DESCRIPTION - Use of a plasminogen activator for manufacturing a medicament for increasing an effect caused by an IL-2 (interleukin-2) inhibitor is new.

INDEPENDENT CLAIMS are included for:

(1) a composition comprising a **plasminogen activator** and **IL-2 inhibitor** as a combined preparation for simultaneous, separate or sequential use for neuroprotective activity;

(2) a manufactured article comprising packaging material containing a plasminogen activator and comprising a label or written material which indicates that the **plasminogen activator** can be used for increasing an effect caused by **IL-2 inhibitor**; and

(3) a manufactured article comprising packaging material containing an IL-2 inhibitor and comprising a label or written material which indicates that the IL-2 inhibitor can be used for increasing or decreasing an effect caused by a plasminogen activator.

ACTIVITY - Neuroprotective; hemostatic; vasotropic; cerebroprotective; cardiant; thrombolytic; nootropic; anticonvulsant; antiparkinsonian.

Thrombolytic occlusion of the MCA (not defined) was induced in rats and two hours later the **IL-2 inhibitor** tacrolimus (FK506) and a tissue-type **plasminogen activator** (t-PA) were administered both alone and in combination. Alone, FK506 and t-PA showed a relatively small inhibition of brain damage, however the combination of FK506 and t-PA caused significant reduction of ischemic brain damage and produced an inhibition value of 23% (greater than that of FK506 or t-PA alone). Additionally, when the drugs

were administered 3 hours after occlusion of the MCA, t-PA was found to increase the level of brain damage (-13.8 plus or minus 7.0%). In contrast, the combination of FK506 and t-PA caused a significant reduction of ischemic brain damage and produced an inhibition value of 16.2 plus or minus 7.6%. These results show that FK506 is able to decrease the serious damage caused by t-PA.

MECHANISM OF ACTION - The IL-2 inhibitor inhibits the production and activity of IL-2 (claimed). The inhibitor inhibits the transmission of the IL-2 signal.

USE - The **plasminogen activator** and **IL-2 inhibitor** in combination are useful for treating acute or chronic cerebral neurodegenerative diseases, e.g. cerebral ischemic diseases and/or brain damage caused by ischemia (including cerebral infarction, head injury, hemorrhage in the brain such as subarachnoid hemorrhage or intracerebral hemorrhage, cerebral thrombosis, cerebral embolism, cardiac arrest, stroke (such as acute stroke), transient ischemic attacks, hypertensive encephalopathy, Alzheimer's disease, Huntington's disease, Parkinson's disease and amyotrophic lateral sclerosis (ALS). The **IL-2 inhibitor** is useful for increasing or decreasing an effect caused by **plasminogen activator** which comprises a neuroprotective activity or brain damage appeared in case that plasminogen activator is administered after its proper therapeutic time (sic).

ADVANTAGE - Administration of **IL-2 inhibitors** and **plasminogen activators** in combination prolongs the therapeutic time window and also produces increased efficiency and safety in the treatment of ischemic brain damage and administration of an **IL-2 inhibitor** decreases the serious damage caused by a **plasminogen activator**.

Dwg.0/0

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=> d l4 4 bib ab

L4 ANSWER 4 OF 5 USPATFULL on STN
AN 2004:4504 USPATFULL
TI Tumor necrosis factor receptor 2
IN Stanton, Jr., Vincent P., Belmont, MA, United States
PA Nuvelo, Inc., Sunnyvale, CA, United States (U.S. corporation)
PI US 6673908 B1 20040106
AI US 2001-968455 20011001 (9)
RLI Division of Ser. No. US 2000-649035, filed on 25 Aug 2000 Continuation-in-part of Ser. No. US 2000-590749, filed on 8 Jun 2000, now abandoned Continuation-in-part of Ser. No. US 2000-495780, filed on 1 Feb 2000, now abandoned Continuation-in-part of Ser. No. US 2000-492712, filed on 27 Jan 2000, now abandoned Continuation-in-part of Ser. No. WO 2000-US1392, filed on 20 Jan 2000 Continuation-in-part of Ser. No. US 968455 Continuation-in-part of Ser. No. US 1999-451252, filed on 29 Nov 1999, now abandoned Continuation-in-part of Ser. No. US 1999-427835, filed on 26 Oct 1999, now abandoned Continuation-in-part of Ser. No. US 1999-414330, filed on 6 Oct 1999, now abandoned Continuation-in-part of Ser. No. US 1999-389993, filed on 3 Sep 1999, now abandoned Continuation-in-part of Ser. No. US 1999-370841, filed on 9 Aug 1999, now abandoned Continuation-in-part of Ser. No. US 1999-300747, filed on 26 Apr 1999, now abandoned
PRAI US 1999-131334P 19990426 (60)
US 1999-131191P 19990426 (60)
US 1999-121047P 19990222 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Benzion, Gary; Assistant Examiner: Chakrabarti, Arun Kr.
LREP Fish & Richardson P.C.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 0 Drawing Figure(s); 0 Drawing Page(s)
LN.CNT 17463
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present disclosure describes the use of genetic variance information for genes involved in inflammatory or immunologic disease, disorder, or dysfunction. The variance information is indicative of the expected response of a patient to a method of treatment. Methods of determining relevant variance information and additional methods of using such variance information are also described.

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